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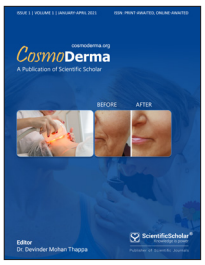

Review Article

Unraveling the diet-dermatitis connection: A systematic review of dietary exclusion in children with atopic dermatitis

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ABSTRACT

This review focuses on atopic dermatitis (AD) impacting 10–30% of children and influenced by food and aeroallergens. It highlights the need for cautious interpretation of sensitization to foods, as it does not guarantee allergy and the potential harm of indiscriminate avoidance affecting nutrient intake. Tests such as skin prick tests, serum-specific immunoglobulin E tests, and food challenges are essential before implementing food elimination. The objective is to assess current dietary exclusion practices in AD children. By screening eight studies including randomized controlled trials, systematic reviews, and cohort studies, the review underscores the benefits of evidence-based dietary exclusion for moderate to severe cases. It establishes causal links between food allergy (FA) and AD emphasizing evidence-based dietary modification over blanket elimination. Temporal changes in allergen antigenicity also suggest evidence-based, supervised practices. In conclusion, the review supports the benefits of evidence-guided food exclusion for AD with 50% of studies backing moderate-to-severe cases. It highlights the need for robust trials to guide rational food restriction, ultimately aiding patients and parents.

Keywords: Atopic eczema, Atopic dermatitis, Food allergy, Dietary exclusion, Dietary modification, Food sensitization

INTRODUCTION

Atopic dermatitis (AD)

The AD is a chronic, pruritic, and inflammatory condition typically initiating before age two, often referred to as atopic eczema (AE).[1,2] Atopy, marked by a predisposition to produce immunoglobulin E (IgE) in response to allergens, is common in eczema patients, leading to the term AE. Not all eczema cases show elevated IgE prompting the use of "atopic eczema," and distinguishing non-IgE mediated eczema.[3] The AD is a prevalent allergic condition, affecting 10–30% of children, commonly triggered by food and aeroallergens in atopic individuals.[4] It stands as the most common childhood inflammatory skin disease in developed countries.[5]

The etiology of AD is multifactorial involving interactions between environmental, immune, and genetic factors. Environmental factors may drive AD through an inflammatory response to allergens (Inside-outside theory) or barrier dysfunction (Outside-Inside theory). Filaggrin gene mutation leads to skin barrier defects, causing dryness, and increasing susceptibility to

sensitization.[1,6-8] Dry skin is a hallmark, emphasizing its importance in diagnosis.[8]

NICE guidelines in 2021 diagnose AE in children based on an itchy skin condition with specific criteria, including a history of flexural dermatitis, visible flexural dermatitis, dry skin in the first 12 months, onset before age 2, and a family history of asthma, allergic rhinitis, or AD.[9] The clinical features of AD include dryness, papules, itching, crusted eczematous areas, Lichenification, and pigmentation changes. It is classified by severity (mild, moderate, and severe) and phases (infantile, childhood, and adulthood) based on age.[10,11] Identifying triggers is crucial in managing AE. Food and environmental factors such as allergens, irritants, and infections can exacerbate symptoms. Topical corticosteroids are first-line treatments for flares.[12]

Management varies by severity. Mild cases use emollients and mild corticosteroids. Moderate cases may include moderate potent corticosteroids, calcineurin inhibitors, and bandages. Severe cases involve potent corticosteroids, calcineurin inhibitors, bandages, systemic corticosteroids, and phototherapy. Biologics such as dupilumab and Janus kinase inhibitors such as baricitinib, abrocitinib, and upadacitinib are approved for moderate to severe cases.[13,14]

Food allergy

Food allergy (FA) constitutes 20–40% of AD and is a recognized public health issue.[5,15] It encompasses both non-IgE-mediated and IgE-mediated reactions with a global prevalence of 10% affecting more children than adults.[16] Food sensitization or intolerance involves difficulty digesting specific foods causing abdominal discomfort, while food allergy triggers immunological reactions impacting multiple organs and may lead to life-threatening conditions as described by Das and Panda.[17]

Food hypersensitivity plays a role in the development of AD,[18] with uncertain significance in children as noted by Eller *et al.*[19] Early sensitization and atopic predisposition influence the onset. Sensitization does not necessarily imply allergy; therefore, comprehensive assessments including skin prick tests, serum-specific IgE, and food challenges are vital before recommending food elimination.[5,15,20,21] Blanket food elimination risks nutritional deficiency and reduced quality of life with only 51% of suspected food allergy cases consulting a dietician.[21] The role of diet exclusion in managing AE in young children is uncertain,[22] and prolonged diet elimination can lead to loss of tolerance and development of food allergy.[21] A study done in the UK, children with food allergies, who had food elimination of more than three foods, found them to be more underweight compared to the general population.[23] Geographical variations in food allergy, highlighted by Genuneit *et al.*,[16] emphasize the importance of identifying specific allergens before eliminating foods.

Clinical presentations of food allergies affecting various body systems, with skin manifestations being predominant, are illustrated in Table 1. Food allergy symptoms range from mild-to-severe life-threatening reactions including anaphylaxis.[16]

The gold standard test for food allergy is the double-blinded placebo-controlled oral food challenge, considered the most reliable diagnostic method.[20,24,25] Despite its efficacy, it carries a risk of allergic reactions and should be conducted in a controlled environment.[25] Additional tests such as skin prick tests and serum-specific IgE tests, can support diagnosis by identifying specific antibodies and sensitization to allergens, though they may not always correlate with clinical reactions due to higher sensitivity and poor specificity.[16] Over interpretation of allergy tests can lead to unnecessary food avoidance emphasizing the need for careful assessment.[20,25] Managing food allergy involves identifying and isolating triggering foods personalized to the patient's clinical presentation.[26] Dietary elimination should align with nutritional requirements with periodic evaluations to assess food tolerance.[27] De Martinis *et al.*[28] recommend supervised use of modified foods such as baked milk, eggs or peanuts rather than complete avoidance.

Treatment options include antihistamines, topical corticosteroids, nasal sprays, and adrenaline in emergencies like anaphylaxis.[26] Allergen-specific immunotherapy, both oral and sublingual, has been explored for desensitization and tolerance induction with varying outcomes.[29-31] Oral immunotherapy is recommended for persistent allergies in children aged four to five years, and biologics like omalizumab are suggested, while reslizumab and mepolizumab are still under investigation.[28]

AD and food allergy

Approximately one-third of children with AD concurrently develop food allergies, and epithelial barrier dysfunction plays a pivotal role in both conditions.[7] As described by Bolognia *et al.*,[10] AD is a complex genetic disorder, is often accompanied by rhinoconjunctivitis, food allergy, asthma, and less frequent esophagitis.

Table 1: Clinical presentation of food allergies.

System affected	Clinical presentation.
Skin	Erythema, urticaria, and angioedema.
Gastrointestinal	Nausea, vomiting, diarrhea, and abdominal pain
Respiratory	Rhinorrhea, bronchospasm, edema of the larynx, and nasal obstruction.

The allergen exposure hypothesis, as proposed by Flinterman *et al*.,[32] suggests that cutaneous exposure to food allergens induces allergic sensitization, while early oral consumption of food proteins may foster immune tolerance. This theory is supported by De Martinis *et al*.,[28] who argue that reduced exposure to infections and microorganisms in early childhood leads to an immune response imbalance favoring a Th2 lymphocyte profile and the production of proinflammatory cytokines like interleukin (IL)-4 over the Th1 lymphocyte profile.

Food allergy is more prevalent in children, and suspicion should arise in infants with moderate-or-severe AE unresponsive to optimal management, particularly when accompanied by failure to thrive or gut dysmotility.[33] Hon *et al*.[34] noted that parents of children with AD often resort to food avoidance strategies, restricting or eliminating certain foods, commonly beef and seafood, to alleviate symptoms.

Common allergenic foods in children include hen's eggs, cow's milk, peanuts, and tree nuts (cashew nuts and hazelnuts) with symptomatology varying based on IgE-mediated or non-IgE-mediated reactions. The IgE-mediated food allergies manifest immediately while non-IgE-mediated reactions occur several hours or days after ingestion. Healthcare providers should be vigilant about the heightened risk of food allergies in infants with severe AE, especially if the onset is before three months of age.[35,36]

Pathophysiology of AD

The AD is attributed to a defective skin barrier and immune dysregulation driving cell-mediated cytokine-driven inflammation.[1,36] Trikamjee *et al*.[36] and Peng and Novak[37] elucidated the intricate interplay among defective skin barriers, nutritional factors, environmental influences, and immune dysregulation in early life. Epidermal barrier dysfunction allows easier entry of microbes, allergens, and irritants triggering the immune system and releasing proinflammatory cytokines impairing the barrier and causing transepidermal water loss[10] [Figure 1]. A major contributor to this barrier dysfunction is the filaggrin gene mutation located at the epidermal differentiation complex in chromosome 1, which decreases filaggrin expression essential for normal skin barrier functions. Filaggrin deficiency correlates with increased transepidermal water loss, reflecting AD severity.[6]

Filaggrin loss represents a genetic risk for AD implicating epidermal dysfunction and subsequent TH2 immune response.[10] The World Allergy Association noted that immune system-dependent epidermal barrier function involves Th2 cytokines, like IL-4, inhibiting S100 and filaggrin expression further impairing the barrier. The Th2 cytokines, particularly IL-4 and IL-13, contribute to acute and chronic AD lesions suppressing epidermal differentiation and antimicrobial peptide production.[6]

Physical skin irritation such as sweating, UV exposure, hot water use or mechanical damage from scratching exacerbates epidermal barrier impairment leading to increased transepidermal water loss and skin dryness.[37] This emphasizes that acquired skin barrier dysfunction can result from mechanical disruption or exposure to irritants triggering inflammation through thymic stromal lymphopoietin release.[6]

MATERIALS AND METHODS

The primary aim of this study was to critically examine current practices in dietary exclusion for children with AD, offering insights into rational dietary modifications and the intricate role of food in this dermatological condition. Through a comprehensive review, the research seeks to contribute valuable insights for more informed and effective dietary interventions in pediatric cases of AD.

A systematic review was conducted using available literature fulfilling the inclusion criteria from the year 2009 to 2023 to assess the current practices on dietary exclusion in children with AE. This review focused on the practices used in dietary exclusion given food hypersensitivity, food as a trigger to AE, and the coexistence of food allergy in children with AD.

The following terms/keywords syntax a search of studies was undertaken: "*Atopic eczema*," "*atopic dermatitis and food allergy*," "*dietary exclusion and eczema*," "*dietary exclusion and atopic dermatitis*," "*dietary modification and eczema*," "*food sensitization and eczema*," "*food sensitization and atopic dermatitis*," and "*food and eczema*."

The following literature search engines were used such as EBSCO *host* using MEDLINE database, Cochrane, PubMed, and Google Scholar. An individual literature search was also done using EMBASE for some of the literature, which was missing the full articles, which when manually added to the Covidence software was noted as duplicate.

Inclusion and exclusion criteria

The inclusion and exclusion criteria used in this systematic review are summarized in Table 2.

Study screening and data extraction

Through the inclusion and exclusion criteria, the articles that merit critical appraisal from all identified citations stored in the electronic databases were selected. The screening of studies was done using the Covidence software, a screening

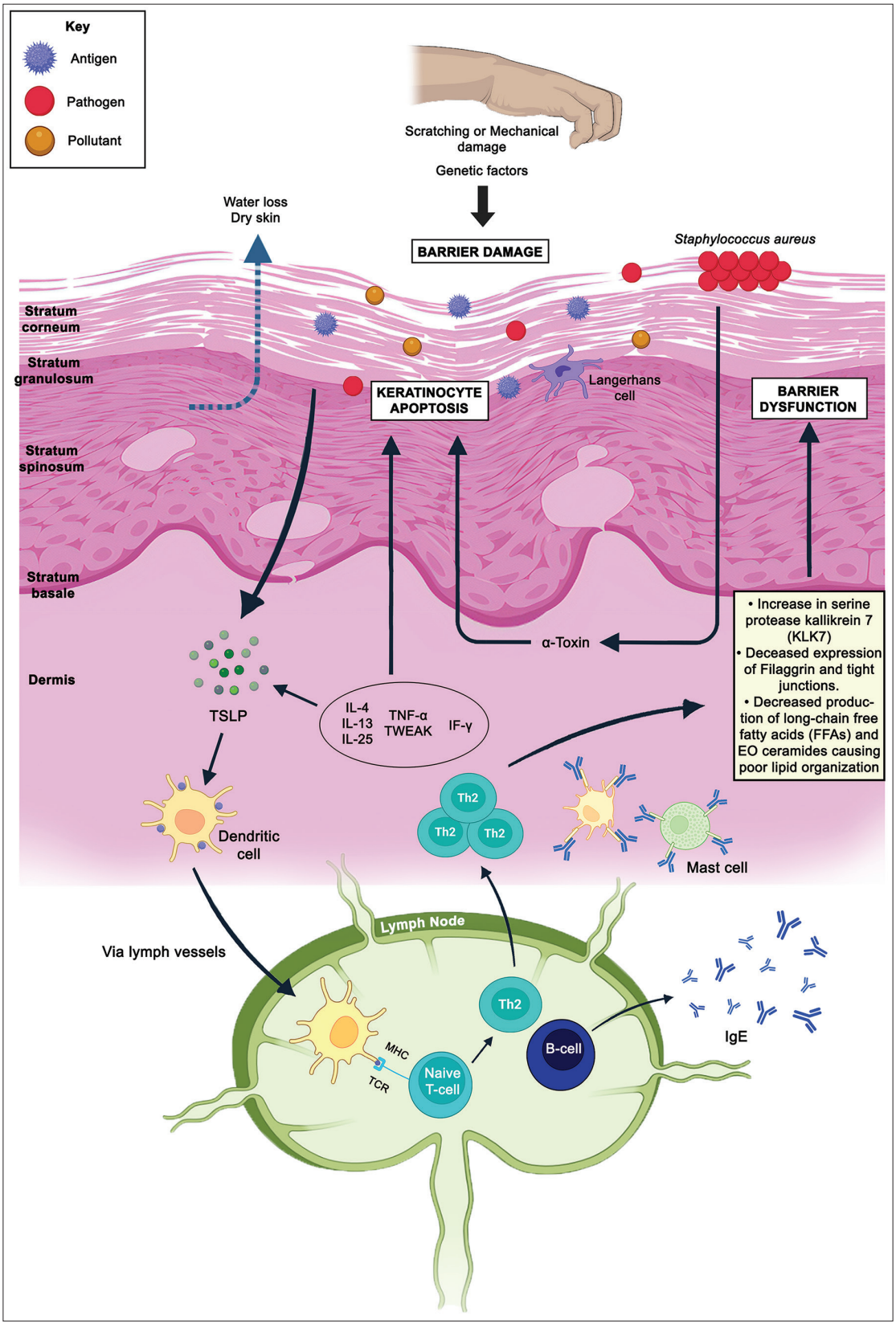

Figure 1: Effects of skin barrier on the pathogenesis of atopic dermatitis. IgE: Immunoglobulin E, Th2: T helper 2, IL: Interleukin, TSLP: Thymic stromal lymphopoietin, TCR: T-cell receptors, MHC: Major histocompatibility test, TNF: Tumor necrosis factor, IF: Interferon, TWEAK: Tumor necrosis factor-like weak inducer of apoptosis.

Table 2: Inclusion and exclusion criteria.

Inclusion criteria	Exclusion criteria
Data from 2009 to 2023.	Data obtained before 2009.
All types of studies including grey literature looking at practices of dietary exclusion in children with atopic eczema.	Articles in which full text was not accessed
Data involving children (<18 years) with confirmed diagnosis of atopic dermatitis.	Data/articles that were not written in English.
	Studies that involved food allergy/sensitization, eczema and other condition/s.

and data extraction tool for conducting systematic reviews, which allows efficient and easily tracked screening. Using this software, 439 searched literatures from EBSCO *host* using MEDLINE database, Cochrane, PubMed, and Google Scholar were imported to the software where seven duplicates were removed. The screening of the titles and abstract was done for 432 citations. Among the 432 citations title and abstract, screening 56 studies warranted their full-text articles review to provide adequate information whereas further screening excluded 40 citations due to different reasons as shown in the PRISMA diagram below [Figure 2] and ended up with 16 citations.

Among the 16 citations, eight citations were then manually excluded (five were reviews, one each were a case report, a commentary, and a symposium article, which were not eligible for critical appraisal). Therefore, eight studies met the inclusion criteria and were used in this review where three studies were randomized controlled studies, three studies were systematic reviews, and two studies were cohort studies.

Quality assessment of the eight studies included in this review was done where through Covidence software; three randomized clinical trials were assessed using Cochrane risk of bias as shown in Table 3.

Assessment of the methodological quality for three systematic reviews included in this review was done using the AMSTAR-2 tool, which is a 16-item tool that is reported to be effective for assessing the quality of systematic reviews that include non-randomized, randomized studies of a health intervention or both, and it is rated as high, moderate, low, and critically low quality as shown in Table 4.

The Newcastle-Ottawa scale was used to assess the quality of two cohort studies used in this review [Table 5].

Data extraction

The process of data extraction involved gathering pertinent information from the included studies to address specific research questions. First, the investigation sought to understand the rationale behind implementing dietary exclusion in patients with AE. Second, it aimed to ascertain whether the decision to undertake dietary exclusion in these patients was evidence based. Finally, the inquiry focused on identifying the frequently excluded foods in individuals with AE or dermatitis. This systematic approach to data extraction aimed to provide a comprehensive understanding of the motivations, evidence basis, and common practices associated with dietary exclusion in the context of AE.

RESULTS

To ensure transparency of the literature used in this systematic review, Table 6 has summarized this.

DISCUSSION

Through a comprehensive literature search, five databases were searched with a high yield in PubMed (278) followed by Cochrane (80) as seen in the PRISMA diagram and the articles were excluded for duplication. Eight studies met the eligibility criteria in this systematic review where three were randomized controlled trials (RCTs), three were systematic reviews, and two were cohort studies.[38-45] Conflicts of interest were reported in 50% of the studies used in this review. Due to differences in quality and study designs of studies that met the inclusion criteria and increasing heterogeneity, a meta-analysis could not be performed.[41] When assessing the severity of AD in studies that were used in this systematic review, different scoring system was used such as SCORAD, POEM, and EASI. In young infants, AD is reported to be the main risk for food sensitization, and this is due to the defective skin barrier, which predisposes to epicutaneous sensitization of food allergens.[8] In this systematic review, the main reason for dietary exclusion in children with eczema was to alleviate the eczema symptoms where parents of children with eczema had tendencies to do dietary elimination, and some of them were advised from health-care facilities. Notably, Palmer *et al.*[39] revealed the reason for dietary elimination in children with AD to be the prevention of food allergy. This was also stressed by Nosrati *et al.*[45] where among the study participants, 87% reported following dietary elimination of different types of foods with an emphasis on empowering the patients on the role of food in the management of AE and the benefits of diet elimination since only 32.5% of patients visited a dermatologist. In their study, there were variations in the duration of dietary exclusion ranging from three weeks to six weeks.

Nearly, all the studies in this systematic review used patients with moderate-to-severe AE except a study that was done by Ridd *et al.*[38] where study participants had mild-to-severe forms of eczema. Children with moderate-to-severe eczema or refractory eczema have a risk of developing food allergy,

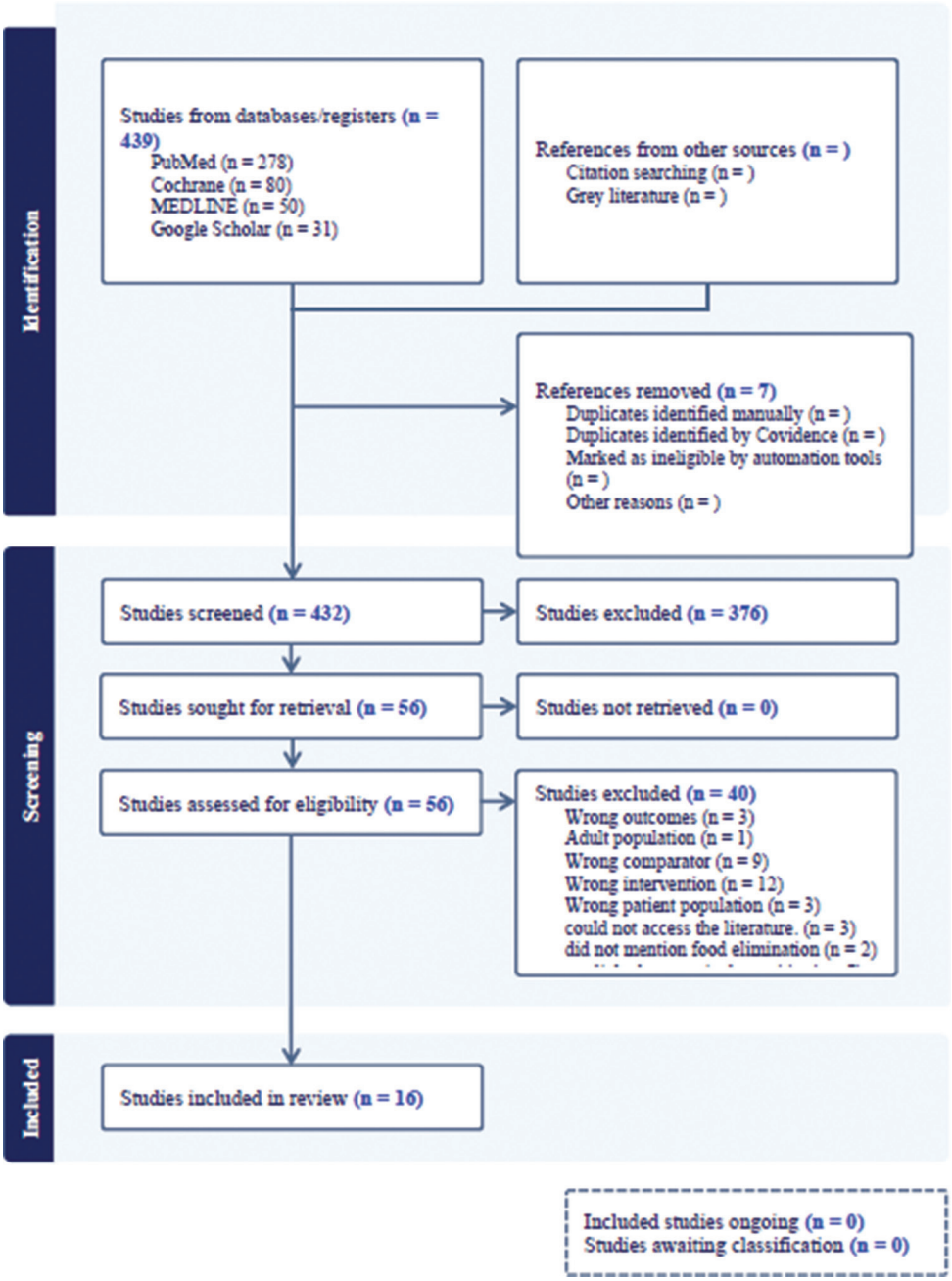

Figure 2: Schematic flowchart of the study.

Table 3: Cochrane risk of bias for three randomized clinical trials.

Study	Allocation concealment	Blinding of outcome assessor for all outcomes	Blinding of outcome for all outcomes	Other sources of bias	Incomplete outcome data for all outcomes	Selective outcome reporting	Sequence generation
Mathew *et al.*[8]	Unclear	Unclear	High	Unclear	Unclear	Low	Unclear
Ridd *et al.*[38]	Unclear	Unclear	Unclear	Unclear	Low	Low	Low
Palmer *et al.*[39]	Low	Low	Low	Unclear	Unclear	Low	Low

and hence, the recommendation is that they should be investigated for food allergy especially when eczema starts early in infancy (<3 months). However, mild cases of AD may not have such a risk.[33] This further made it difficult to provide a generalization of risk since most of the studies used moderate-to-severe eczema, which is reportedly a risk of developing food allergy.

Table 4: Quality assessment of included systematic reviews using the AMSTAR-2 tool.

Studies	Tsakok *et al.*,[42] 2016	Roberts *et al.*,[43] 2022	Das and Panda,[17] 2021
Q.1	Yes	Yes	Yes
Q.2	Yes	Yes	Partial yes
Q.3	Yes	Yes	No
Q.4	Yes	Partial yes	Yes
Q.5	Yes	Yes	Yes
Q.6	Yes	Yes	Yes
Q.7	Yes	Yes	Yes
Q.8	Yes	Yes	Partial yes
Q.9	Yes	Yes	No
Q.10	No	Yes	Yes
Q11	Yes	No meta-analysis conducted.	No meta-analysis conducted.
Q.12	Yes	No meta-analysis conducted.	No meta-analysis conducted.
Q.13	Yes	Yes	Yes
Q.14	Yes	Yes	No
Q.15	No	Yes	No meta-analysis conducted.
Q.16	Yes	Yes	Yes
Conclusion	Low	High	Critical low

In this systematic review, foods that were excluded were cow's milk, hen's egg, peanut, codfish, rice, prawns, soybeans, seafood, and wheat. When a tailored elimination diet guided by a good history and confirmatory allergy tests is recommended, it helps to give a practical and rational dietary freedom to the patient, hence the importance of identifying the definite allergen instead of a blanket elimination.[17] This is of paramount importance especially in children since an inadequate nutrient replacement following food elimination can result in nutritional deficiencies. As an example, children under two years of age with cow's milk allergy developed protein energy malnutrition following the use of rice milk as a substitute for cow's milk because rice milk could not meet the nutritional demands of these children under two years in this critical period for their growth and development.[44,46] Paradoxically, most children develop food allergies <2 years of age; thus, a prompt action in diagnosing a specific food allergy is important since unnecessary food avoidance can put children at risk of nutrition deficiencies and failure to thrive.[47]

Most parents suspect that all eczema symptoms are due to food allergies and implicate their role in the flares of AE. They often seek an allergy test to specifically guide them in their judicious dietary decision including dietary

Table 5: Newcastle-Ottawa scale was used to assess the quality of two cohort studies.

	Selection	Score	Comparability	Score	Outcome	Score	Risk of bias.
Eller *et al.*[19]	1. a) Truly representative.	1	1.a) The study controls for age, sex and marital status.	1	1.b) Record linkage	1	Good quality study-Low risk of bias
	2. b) Drawn from the same exposed cohort	1			2.Yes	1	
	3. b) Structured interview.	1			3.b) Subject loss to follow up unlikely to introduce bias-number lost≤20% or description of those lost suggested no difference no different from those followed.	1	
	4.Yes	1					
Dhar *et al.*[44]	1.a) Truly representative	1	1.c) Cohort are not comparable on the basis of the design or analysis controlled for confounders.	0	1.b) Record linkage	1	Poor quality-High risk of bias.
	2.c) No description	0			2.a) Yes	1	
	3.a) Secure records	1			3.d) No statement.	0	
	4.Yes	1					

Table 6: Literature used in this systematic review.

Author/s and year of publication	Date searched	Sample size	Age range	AD severity	Food challenge done	Specific IgE test	Excluded food/s.
Mathew *et al.*[8]	19/3/2023	30	1 year–15 years	Not stated.	Yes	No	Cow's milk and eggs
Ridd *et al.*[38]	22/3/2023	80	3 mo–<5 years.	Mild-severe AD	Yes	Yes	Cow's milk, peanut, hen's egg, codfish
Palmer *et al.*[39]	20/3/2023	86	4–8 mo	Moderate to severe AD	Yes	IgE/IgG4	Eggs, rice
Tsakok *et al.*[42]	19/3/2023	Not stated	Multiple studies-	Not stated	Yes	Yes	The individual study specified.
Roberts *et al.*[43]	20/3/2023	1st RCT-62 2nd RCT 84	1st RCT 11–17 mo 2nd RCT 3 mo–5 years	1st RCT 11–17 mo. -moderate to severe. 2nd RCT 3 mo–5 years- Mild to severe eczema.	Yes	Yes	Cow's milk, peanut, hen's egg, codfish
Das and Panda[17]	20/3/2023	N/A	Infants and young children	Not specified	Yes	Yes	Eggs, cow's milk, food preservatives, gluten, food colorings, sugar, nightshades, and food additives.
Dhar *et al.*[44]	21/3/2023	100	6 mo–12 years	Not specified	Yes	Yes	Eggs, milk, nuts, prawns, soybeans, and seafood
Eller *et al.*[19]	20/3/2023	562	0 mo–6 years	Mild to severe AD.	Yes	Yes	Eggs, milk, codfish, eggs, wheat, and peanut

IgE: Immunoglobulin E, AD: Atopic dermatitis, RCT: Randomized controlled trial

eliminations.[38,46-51] Tait and Goldman,[48] however, stressed that the parents' report of eczematous reactions is sometimes a poor indicator of clinically relevant food allergy; hence, the emphasis should be on coupling the history, sensitivity tests, and confirming with the gold standard test for diagnosing specific food allergy. This is in keeping with what Tsakok *et al.*[42] who postulated that food sensitization is likely to result from an inflamed skin barrier with the severity of eczema reported as a major risk factor for sensitization at an early age. This could support the fact that AD is thought to precede the development of food allergy due to epicutaneous sensitization caused by defective skin barrier in AE.[8]

Approximately 80% of children with AD will have elevated food/allergen-specific IgE, but 35–40% will have confirmed food allergy.[17] This is in keeping with a study done in South Africa by Gray *et al.*[49] where they found that among 100 children with AD, 60% were sensitized to food/s, but only 40% had proven food allergy. Furthermore, Das and Panda[17] emphasized that avoidance of proven food allergens can be practiced in the management of children with AD especially those who were previously sensitized. However, it should be guided, supervised, and must take into account the nutritional needs of the patient.

Delay in introducing allergenic foods such as oats, cow's milk, fish, and eggs beyond six and nine months has been associated with an increased risk of allergenic diseases.[39] Early introduction of such food is reported to promote food tolerance in young children; thus, parents and patients need to be educated about the effect of irrational avoidance of food since it can cause loss of food tolerance and cause subsequent food-induced immediate hypersensitivity reaction and anaphylaxis.[17,24,49-51] An Australian health nut study revealed a significant risk of egg allergy in children, who were introduced to eggs between the age of 10–12 months or more when compared to those who were introduced at the age of four to six months of age.[39]

Avoidance of food should be based on the history of exacerbation of symptoms and confirmed diagnosis of food allergy. When there is a consistent correlational symptom, a diagnostic elimination of up to 4–6 weeks can be done, and

after an improvement of symptoms after dietary elimination, it should be confirmed by a double-blinded placebo control food challenge.[8] Compared with IgE-mediated food allergy, which occurs immediately, non-IgE-mediated food allergies pose challenges due to the time delay between food contact and symptom (which normally takes 48–72 h).[52] In such cases, no identifying test can be done, so the only diagnostic test will be to supervise dietary elimination of the suspected food allergen for four weeks expecting partial or complete resolution of symptoms such that the patient may present with abdominal cramps, vomiting, colic, diarrhea, failure to thrive, and blood in the stool.

Egg allergy and cow's milk allergy are reported to be common in the younger age group when compared with fish allergy, tree nuts, and peanut allergy, which are reported to be common in older age groups.[38,53] Furthermore, Ridd *et al.*[38] reported that allergic reactions to different types of food may cause eczema symptoms and eczema is associated with food allergy. Tsakok *et al.*[42] emphasized that 53–66% of people with eczema are food sensitized, while 15–81% report challenge-proven food allergy; this was also reiterated by Gray *et al.*[49] who further emphasized that being sensitized to food does not necessarily mean allergic. There is a reported causal relationship between food allergy and AD, patients with AD were reported to be six times more likely to develop food sensitization when compared with their healthy controls.[42] Severe persistent forms of AD and the early onset of AD are reported risk factors for food allergy.[38,39,49] Early introduction of food has shown some benefits in developing oral tolerance.[38,49] The defective barrier function in AD could explain the sensitization to food, which emphasizes the likelihood that AD is involved in the pathogenesis of food allergy rather than food exacerbating the AE, as epidermal barrier dysfunction is significant in the pathogenesis of AD.[17,48,54] The blanket elimination of cows' milk and eggs in all children with AE is injudicious; it was reported to be only beneficial in those patients, who were sensitized or allergic to cows' milk and egg.[48,54] Furthermore, Roberts *et al.*[43] emphasized the rational use of dietary modification in children with AD since it can lead to long-term effects through malnutrition, discouragement of breastfeeding, and loss of oral tolerance to excluded foods. Unnecessary avoidance of food can cause a detrimental impact on the quality of life in patients with food allergies (FA).[15,50] A diagnosis of FA is reported to have serious implications in patients' life, limiting social activities and dietary freedom. Therefore, it is important to have a proper diagnosis since misdiagnosis can put a patient at risk of developing severe allergic reactions.[25] A study done by Marklund *et al.*[21] revealed that children, who were excluded from more than two diets were shorter compared to those who eliminated one diet. This shows that blanket dietary elimination can impair the growth and well-being of children. A study done among children aged 1–22 months in the US reported cases of kwashiorkor following dietary elimination, the main reasons being nutritional ignorance, food faddism or perceived milk intolerance.[55] Tait and Goldman[48] and Eigenmann *et al.*,[50] also supported that the elimination of diet can jeopardize the nutritional status, and contribute to the development of clinical food allergy and failure to thrive. A study done in South Africa by Gray *et al.*[49] revealed a high prevalence of food sensitization which was comparable with mixed races; however, the proven food allergy was less when compared to mixed races, and a significantly low peanut allergy in black children. This emphasizes the importance of test-guided dietary modification, instead of doing blanket or blind elimination of food/s; also, some of the allergens may be outgrown as children grow; therefore, it is important to be evidenced based and supervised and emphasize the proper interpretation of sensitivity tests since being sensitized does not necessarily mean allergic. Roberts *et al.*[43] elaborated that there is a variation in healthcare practices on dietary elimination in established eczema. Ridd *et al.*[38] emphasized the importance of clinicians focusing on promoting control of eczema and not cure messages when communicating with parents/patients by ensuring they emphasize the use of moisturizer, topical, and systemic medications as indicated. It is also important for a healthcare provider to explain to the caretaker/patient the relapsing nature of this chronic and relapsing inflammatory condition by avoiding the possible triggers and aiming at alleviating the presenting symptom instead of thinking of a cure. This was echoed by Eigenmann *et al.*,[50] who emphasized the proper management of AE and looking for other possible triggers for AD since there are other triggers for eczema apart from food allergens. Only a third of patients with moderate-to-severe eczema are at risk of developing food allergy, and for those who are suspected to be allergic limited panel of food tests can be performed while explaining to the patient/parents the pros and cons of dietary elimination. Ensuring proper management of AD was also highlighted by Olabi and William[2] who reported that underuse of topical therapy is the primary cause of treatment failure in AD, and this is chiefly due to the fear of toxicity of medications such as steroids. The AD is common in children; thus, the attending clinicians will regularly encounter children with food allergies or suspected FA; hence, the importance of educating healthcare providers to do a proper diagnosis of FA and rational avoidance of food, which is guided by proper investigation.[52] Mehta *et al.*,[47] further, stressed on ensuring proper dietary interventions for children with restricted foods due to food allergies to ensure the provision of adequate nutrients to avoid detrimental effects such as growth retardation. This systematic review provides a valuable opportunity for advancing the discourse surrounding the dietary restrictions in patients with AE emphasizing the need for timely medical intervention, and recognizing their profound relevance in effectively managing this prevalent childhood condition on a global scale.

CONCLUSION

This systematic review has unveiled a range of practices surrounding dietary exclusion in children diagnosed with AE revealing a common inclination among both parents and healthcare providers to eliminate specific foods due to concerns over exacerbating eczema symptoms. Parents often seek food allergy tests to guide their decisions regarding dietary modifications. It is important to educate parents and patients about the potential consequences of food avoidance, as it can lead to loss of food tolerance and subsequent immediate hypersensitivity reactions and anaphylaxis. While this review presents varying conclusions due to differences in study designs, a significant number of studies included in this review support evidence-guided food elimination as beneficial for children with moderate-to-severe AD. This emphasizes the need for robust, multi-center RCTs to establish a definitive evidence-based framework, aiding healthcare providers in making judicious decisions regarding food restriction in children with AD.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent was not required as there are no patients in this study.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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